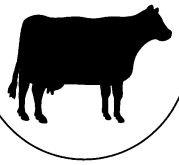

Use of ionophores in lactating dairy cattle: A review

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Abstract — Ionophores are feed additives that alter rumen microbial populations through ion transfer across cell membranes. Although ionophores have been used widely in the beef industry for improved feed efficiency and control of coccidiosis, there has been limited use by the dairy industry. In Canada, the label warning prohibiting the use of monensin premix in lactating dairy cattle was removed in June 1996. Following this, in December 1997, a controlled release capsule containing monensin was approved for use in dairy cattle as an aid to prevent subclinical ketosis. Monensin may have several advantages for dairy cattle, including improved energy metabolism, increased milk production, and altered milk components. This literature review was primarily conducted in 1996 by using the Agricola and CAB search databases. Other relevant articles published since the search (up to 1998) have been added. This review will provide practitioners with relevant references in the published literature regarding ionophore use in dairy cattle. It should also give some guidance as to what effects might be anticipated with the use of ionophores in lactating dairy animals.

Résumé — Utilisation des ionophores chez la vache laitière en lactation : mise au point. Les polyéthers ionophores sont des additifs alimentaires qui modifient les populations microbiennes du rumen par transfert d'ions au travers des membranes. Même si les ionophores ont été largement utilisés dans l'industrie du bœuf de boucherie pour améliorer l'efficacité alimentaire et lutter contre la coccidiose, leur utilisation en industrie laitière est demeurée restreinte. Au Canada, l'étiquette de mise en garde interdisant l'utilisation des prémélanges de monensin chez la vache laitière a été abolie en juin 1996. Par la suite, en décembre 1997, une capsule de monensin à libération contrôlée a été approuvée pour l'utilisation chez la vache laitiére comme aide à la prévention de la cétose subclinique. Le monensin semble posséder plusieurs effets avantageux chez la vache laitière, dont l'amélioration des composantes du lait. Cette revue de littérature a été principalement effectuée en 1996 en utilisant les bases de données Agricola et CAB. D'autres articles pertinents parus depuis (jusqu'en 1998) ont été inclus. Cette revue fournira au praticien des références pertinentes d'articles publiés concernant l'utilisation des ionophores chez la vache laitière ainsi que quelques indications sur les effets anticipés des ionophores chez l'animal en lactation.

(Traduit par docteur André Blouin)

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Introduction

Ionophores have been used extensively in the beef industry in Canada since 1977. Until recently, there has been no label indication for use of ionophores in lactating dairy cattle. In Canada, a monensin controlled release capsule (CRC) is now approved for use in dairy cattle as an aid in the prevention of subclinical ketosis when administered 2 to 4 wk prior to expected calving. Ionophores have been studied regarding several potential effects on lactating dairy cows, including influences on health, milk production, and reproduction. The purpose of this literature review is to examine the basic mode of action of ionophores and the effects of ionophores in lactating dairy cattle.

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Materials and methods

The literature search for this manuscript was conducted by using 2 CD ROM databases called Agricola and CAB. The search was conducted in 1996 by using the following key words: ionophores, lactating, dairy, monensin, and lasalocid. The search included all articles published since 1984. All references identified through this search mechanism were evaluated for proper study design and relevancy to this paper. The majority of studies cited in this review contained good experimental design, including random allocation of individual subjects to treatments and appropriate statistical comparisons. Some recent abstracts (published after 1992) have been cited because the information is useful, given the current relative paucity of data in this area. Several studies were conducted in other countries where management practices and nutrition may not be directly applicable to the Canadian dairy industry; however, these studies were included because they still add

important insight into the effects of ionophores in dairy cattle. Other relevant references published subsequent to the database search (up to 1998) were also included in this review.

Results

Effects of ionophores on ruminant digestion and metabolism

Monensin and lasalocid are the most commonly used feed additives in cattle (1). Monensin is a carboxylic polyether ionophore produced by a naturally occurring strain of *Streptomyces cinnamonensis* (2) and is provided to cattle, PO, as a sodium salt (3). Lasalocid is closely related but produced by a different strain of *Streptomyces*. The basic function of ionophores is to create a flux of ion transport across cell membranes. Monensin binds to bacterial cell membranes and first causes an efflux of potassium from the cell and an influx of hydrogen ions into the cell (4). The increased hydrogen ions are exported out of the cell either by active transport involving adenosine triphosphate or passively via sodium entry into cells in exchange for hydrogen. In order to maintain inner cell equilibrium, the bacterial cell expends energy and this results in death or reduced growth of the bacterium (5). Lasalocid is similar to monensin and also causes ion flux across cell membranes but can translocate both monovalent and divalent cations (5). Since gram-negative bacteria have complex outer cell membranes, they are usually more resistant to the action of ionophores than are gram-positive bacteria. Ionophores, therefore, selectively inhibit gram-positive bacteria rather than gram-negative bacteria, because of differences in bacterial cell wall structure.

Monensin exerts its many effects by shifting the microbial populations in the rumen (5). Three major areas of animal metabolism influenced by monensin were identified (5). These included increased efficiency of energy metabolism, improved nitrogen metabolism, and general digestive effects, including reductions in both bloat and lactic acidosis. Schelling (6) described monensin as having several modes of action, including modified volatile fatty acid production, modified feed intake, changes in gas production, modified feed digestibilities, and alterations in both rumen fill and rate of passage. Monensin changes the ratio of volatile fatty acids in the rumen, increasing propionic acid and reducing the molar percentages of butyric and acetic acid (7). Increased rumen propionic acid improves gluconeogenesis (6). Direct effects on rumen bacteria are probably responsible for the so called protein sparing effect (8,9), and monensin studies in steers and lambs have demonstrated higher circulating urea and lower rumen ammonia levels in treated animals (8,10,11). A reduction in rumen methane production has also been observed with monensin (6). Monensin slightly improves feed digestibility, reduces rumen turnover, and may reduce feed intake, especially on high concentrate diets (6). During the adaptation period, monensin may depress intake and reduce feed digestibility (6). Other effects of monensin include a reduction in 3-methylindole production (6) and a reduction in face fly and horn fly numbers (12). Monensin is also used extensively to help to control bovine coccidiosis (1).

Target animal and human food safety

Several studies have been conducted to measure possible residues of monensin in either milk or tissue of dairy cattle. There is no active monensin in milk at a limit of quantitation of 5 parts per billion (ppb) for doses up to 1274 mg/animal/d in Holstein cows (13,14).

In addition, a number of target animal studies with monensin in cattle have been conducted. An acute tolerance study showed toxic signs in dairy cattle gavaged with 10 mg/kg body weight (BW) (> 5000 mg) of monensin for 4 to 8 d (14). However, at a dose of 1 mg/kg BW (450 to 550 mg), no clinical or pathological evidence of monensin toxicity occurred (13). Signs of monensin toxicity in cattle include acute anorexia, diarrhea, lethargy and weakness, reduced milk production, and dehydration (13). Biochemical abnormalities associated with monensin toxicity include elevated levels of creatinine phosphokinase and aspartate aminotransferase (15).

Potential for use of ionophores in lactating dairy cattle

Improved energy metabolism

The gluconeogenic potential of monensin has attracted researchers to investigate its possible role as an antiketogenic agent in dairy cattle. Rogers and Hope-Cawdery (16) were the first to describe the beneficial effects of monensin for reducing the effects of ketosis in a herd with a clinical ketosis problem. This report was not a controlled randomized trial. The antiketogenic properties of monensin were later investigated in a Canadian trial involving 2 levels of monensin and 3 groups of 12 Holstein cows (17). Monensin included at 30 g/ton of total ration (high group) decreased the incidence of subclinical ketosis and significantly reduced blood beta-hydroxybutyrate (BHB) levels in the first 3 wk postpartum (17). The incidence of subclinical ketosis, defined as total blood ketones > 9 mg/100 mL (900 μmol/L), was decreased and blood BHB levels were reduced by 40% for the high monensin group. The lower monensin dose did not significantly impact blood BHB or subclinical ketosis in this study. Based on the average feed intakes observed in this trial, the low monensin group received approximately 208 mg monensin/animal/day and the high group 399 mg monensin/animal/day (17).

A German study involving 23 German Black and White dairy cows given 240 mg/day of monensin in the ration also reported an antiketogenic effect (18). Animals receiving doses of monensin of 10 and 20 mg/kg in feed commencing before calving and continuing into early lactation showed significant reductions in blood acetone and acetoacetate but no significant effect on BHB (19). Monensin treatment, commencing at 2 to 4 wk prior to calving, reduced serum BHB and nonesterified fatty acids (NEFA) in lactating dairy cows during the first 28 d postpartum, when monensin was fed at 300 or 450 mg/animal/d but not at 150 mg/animal/d (20). Serum glucose was not influenced by feeding monensin.

Several studies involving an intraruminal controlled release capsule (CRC) have been used to evaluate the metabolic, health, and production effects of monensin in dairy cattle. This spring-loaded capsule contains 32 g of monensin in a hexagylcerol distearate matrix core (26).

Table 1. Summary of the metabolic effects of differing doses of monensin in lactating dairy cattle reported from various international studies

Year	Country	*n*	Dose[a]	Ketone bodies	Glucose	Urea	Other	Reference number
1989	Canada	36	16 ppm 33 ppm	↓total ketones ↓BHB (S)[c]	NS	NA	NA	17
1993	South Africa	60	10 ppm 20 ppm	BHB (NS), ↓ACAC (S) ↓milk acetone (S)	NS	NA	NA	18
1993	United States	47	150 mg 300 mg 450 mg	↓BHB (S)[d]	NA	NA	NA	20
1994	Australia	16	CRC	↓BHB (S)	↑(trend)	NS	NEFA (NS)	21
1996	New Zealand	120[b]	CRC	BHB (NS)	NS	↑(S)	NA	22
1997	United Kingdom		300 mg	↓BHB (S)	NA	NA	NA	39
1997	Australia	24	CRC	↓BHB (S) precalving	↓(S) precalving	NS	↓NEFA precalving	25
1997	Canada	1010	CRC	↓BHB (S), ↓milk ketones (S)	↑(S)	↑(S)	↓AST	24
1997	Canada	52	CRC	↓BHB (S)	↑(S)	NS	↑rumen pH (S)	23
1998	The Netherlands	80	300 mg	↓BHB (S), ↓ACAC (S)	↑(S)	NA	insulin (NS)	41

ACAC — acetoacetate; AST — aspartate aminotransferase; BHB — beta-hydroxybutyrate; NA — not applicable; NEFA — nonesterified fatty acid; NS — not statistically significant; S — statistically significant ($P < 0.05$)
[a]Dose: mg = mg/cow/day, CRC = controlled release capsule (335 mg/cow/day), ppm = concentration in mg/kg of total feed
[b]A subsample from each herd was evaluated
[c]only for 33 ppm
[d]only for 300 and 450 mg

In Canadian studies, it has been demonstrated that a CRC containing monensin delivers a constant daily dose of approximately 335 mg for about 95 d in dairy cows (14). Cows in Australia treated with a monensin CRC during the first week postcalving had significantly lower plasma BHB levels and tended to have higher glucose concentrations than did controls receiving no monensin (21). In a New Zealand trial, monensin-treated cows had significantly higher levels of serum urea; however, no significant effects of monensin on glucose or BHB were shown (22). In this study, monensin CRCs were administered 1 mo prior to artificial insemination. This time of administration would likely have been beyond the first 30 d after calving, which is the primary risk period for subclinical ketosis. Therefore, cows in this study were probably not in a negative energy balance during monensin-treatment.

Green (23) reported that administration of a monensin CRC 3 wk prior to expected calving significantly reduced the concentrations of BHB and increased those of glucose. Monensin treatment in this study was also reported to reduce both the onset and severity of subclinical ketosis when cows where restricted to 90% of ad libitum feed intake commencing at 2 wk postcalving. Duffield (24) reported that monensin CRC administration at 3 wk prior to calving reduced the incidence, prevalence, and duration of subclinical ketosis in a 1010-cow multiherd field study. Monensin treatment also significantly reduced the concentrations of serum BHB and aspartate aminotransferase, and increased the concentrations of serum glucose and urea.

Stephenson et al (25) conducted a small study involving 24 cows from 2 farms where monensin CRCs were administered 50 d precalving. A significant decrease in nonesterified fatty acids, BHB, and glucose were noted in the precalving period. No significant effects on these energy indicators were observed postcalving. However, a significant elevation in ceruloplasmin concentration was noted in monensin-treated cows, postcalving. The authors suggested that this increase in copper absorption may assist the cow in fighting oxidative challenges. A summary of monensin's effect on energy metabolism in dairy cattle is reported in Table 1.

Health

A CRC containing monensin has been found to be efficacious for the prevention of pasture bloat in dairy cattle in several studies conducted in Australia and New Zealand (26–28).

Monensin has also been reported to reduce the incidence of subclinical ketosis. Sauer (17) reported a reduction in subclinical ketosis from 6 out of 12 in the untreated group to 4 and 1 out of 12 in the low (208 mg/cow/d) and high (399 mg/cow/d) monensin groups, respectively. This was a relatively small trial conducted in one research herd. The high dose of monensin did significantly reduce the incidence of subclinical ketosis; however, at the low dose, the incidence of subclinical ketosis was not significantly different from that of controls. Duffield (24) reported that monensin delivered in a CRC (335 mg) reduced the incidence of subclinical ketosis by 50%, at threshold values for

defining subclinical ketosis of 1200, 1400, and 2000 μmol/L BHB in 1010 cows from 25 commercial dairy farms. Monensin also significantly reduced the duration of subclinical ketosis.

The use of monensin CRCs precalving has been shown to significantly reduce the risk of abomasal displacement and multiple illness (more than one disease in early lactation) (24). In the same study, there was a tendency for monensin-treated cows to be at reduced risk of clinical ketosis and culling. These health effects were presumably associated with the observed reduced incidence of subclinical ketosis (24). The work of Beckett et al (29) demonstrated no significant health effects of the monensin CRC when it was administered 40 d prior to expected calving; however, disease incidence was substantially lower in this study than would be expected for typical North American dairies.

In feedlot steers, monensin has an impact on reducing rumen acidosis. In one study that measured continuous 24 h rumen pH, monensin reduced the time rumen pH fell below 5.6 and was associated with a more consistent feed intake pattern (30). These effects are thought to be mediated through monensin's effect on reducing lactic acid-fermenting bacteria and enhancing lactic acid utilizers. In the only study to date on the effects of monensin use on rumen pH in dairy cattle, significantly higher rumen pH values were observed, postcalving, in monensin-treated cows (23). However, both placebo (CRC not containing monensin) and monensin groups had point sample rumen pH values well above 6.0. Further work will be needed to investigate the potential of ionophores in preventing rumen acidosis in lactating dairy cattle.

Milk production and milk components

Trials from both Australia and New Zealand have evaluated the effects of monensin on milk production in pasture-fed cattle. A 7% to 8% increase in milk yield over a 14-week period was observed in a single herd study of 90 cows on pasture given a monensin CRC at 46 d postcalving (27). An increase in protein, but not fat, yield was also found. In agreement with these findings, Lowe et al (28) demonstrated increased milk production of 1.1 kg/d (6.2%) associated with monensin treatment in 368 cows from 4 herds that were randomly allocated to a monensin CRC treatment between 0 and 100 d postpartum. In a New Zealand trial involving all the cows from 3 pasture-fed herds, monensin-treated cows produced 0.41 L more milk per day over a 4-month period, and 1.38 L more milk per day than did untreated control cows at the 2nd month after treatment (22). Treated cows also produced slightly less protein (0.006 kg/d) and less fat (0.015 kg/d). Treated cows were administered a monensin CRC, which would have been depleted after the 3rd month, but the effects on production lasted into the 4th month posttreatment. Two other trials involving a monensin CRC treatment reported conflicting results. In one, 16 cows fed ryegrass pasture and 3 kg of dairy supplement were randomized to receive a monensin CRC or no treatment within 48 h of calving (21). No difference in milk yield was found, but milk fat percent was significantly lower in the monensin-treated cows. In the other, involving 1061 lactating cows from 6 different herds, milk fat and milk protein production was not significantly influenced by monensin treatment (31). Milk production was significantly increased in only 1 of the herds. Cows in this trial were randomly assigned to control or monensin CRC treatment within 7 d of calving. Five herds were pasture-fed with supplementary concentrates and the 6th herd was a large dairy herd that was fed a total mixed ration. The herd with the positive milk production response was a pasture-fed herd. The finding of 1 herd with a production effect suggests that there may be herd characteristics (possibly nutritional interactions) that either reduce or enhance the production impact of ionophores. The cows in most of these trials were pasture-fed and the effects discovered may not apply to North American systems. In addition, the treatment evaluated was in all cases a monensin CRC, and it was consistently administered in the first 100 d after parturition.

There have been few studies that evaluated the effect on milk production of the monensin CRC administered precalving. In a large Canadian field study, Duffield (24) found a significant monensin by body condition score (BCS) interaction on milk production. A total of 503 cows were given a monensin CRC and 507 were treated with a placebo capsule at 3 wk prior to expected calving. Cows classified as thin (BCS $\leq$ 3.0) at 3 wk prior to calving had no significant production response to monensin CRC treatment for the first 90 d of lactation. Cows classified to be in good body condition (BCS 3.25 to 3.75) prior to calving had significantly higher milk yield (+ 0.85 kg) at peak lactation, while cows that were considered to be in fat body condition (BCS $\geq$ 4.0) showed a significant production increase of 1.2 kg/d for the first 90 d of lactation. The BCS-monensin interaction may be the result of alleviation of the detrimental impact of subclinical ketosis on milk production, which is more likely in moderate and overconditioned cows. There were no significant effects of monensin treatment on either milk fat or milk protein percentages. However, treatment would have ceased in this trial around 75 d in milk, so there could be impacts of monensin on milk components beyond this stage of lactation. Beckett et al (29) measured milk yield in 915 cows given either a monensin CRC 40 d prior to and 50 d after the expected calving date or were given nothing, and they found an increase in the lactation milk yield of 0.75 kg/d in the monensin-treated cows. This effect was different among the 12 herds in the study. No significant effects of monensin on milk fat or milk protein percentage were reported.

Several studies have investigated the production effects on various concentrations of feed delivered ionophores. Weiss and Amiet (32) found no effect on either milk production or milk components when 340 mg/d of lasalocid was added to the feed of 32 mid-lactation cows. In another trial designed to evaluate the impact of both added fat and lasalocid, reduced fat-corrected milk yield, reduced milk fat percent, and reduced milk fat yield were found associated with inclusion of lasalocid in the ration, commencing at 90 d postpartum (33). Lasalocid was also evaluated at 180 mg/d and 360 mg/d in the diet of 36 early lactation cows. However, no significant effects of the ionophore

Table 2. Summary of the effect of ionophores on milk production and milk components in lactating dairy cattle reported in various international studies

Year	*n*	Ionophore	Dose[a]	DIM at Tx start	Milk response	Fat response	Protein response	Reference number
1988	24	Lasalocid	36.7 ppm	90	NS	↓0.58%	NS	33
1990	90	Monensin	CRC	46	↑1.0 kg/d	↓0.40%	↑0.03 kg/d	27
1990	32	Lasalocid	340 mg	mid lact	NS	NS	NS	32
1993	60	Monensin	10 ppm 20 ppm	-28	↑3.1 kg/d ↑3.4 kg/d	NS	NS	19
1993	36	Lasalocid	180 mg 360 mg	early lact	NS	NS	NS	34
1993	47	Monensin	150 mg 300 mg 450 mg	-28	NS	NS	NS	20
1994	16	Monensin	CRC	0 to 2	NS	↓fat %	NS	21
1994	1061	Monensin	CRC	0 to 7	↑1 herd (out of 6)	NS	NS	31
1995	60	Monensin	150 mg 300 mg 450 mg	42	↑2.8 kg/d ↑2.5 kg/d NS	↓0.33% ↓0.47% ↓0.47%	↓0.15% ↓0.14% ↓0.16%	36
1996	661	Monensin	CRC	1 mo prior to breeding	↑0.41 kg/d	↓fat % and yield	↓protein yield	22
1997	98	Monensin	300 mg	35 — L1 -14 — L2	↑0.8 kg/d ↑1.1 kg/d	↓0.11% ↓0.16%	NS	37[b]
1997	60	Lasalocid	10 ppm 20 ppm	-14	NS	NS	NS	38
1997	1010	Monensin	CRC — thin — fair — fat	-21	NS ↑0.85 kg/d ↑1.2 kg/d	NS	NS	24[c]
1998	64 80	Monensin	450 mg 300 mg	35	NS ↑1.9 kg/d	↓0.41% NS	NS ↑0.06 kg/d	39[d]

DIM — days in milk; Tx — treatment start; lact — lactation; NS = not statistically significant ($P > 0.05$)
[a]Dose: mg = mg/cow/day, CRC = controlled release capsule (335 mg/cow/day), ppm = concentration in mg/kg of total feed
[b]A two lactation study (L1 = 1st year, L2 subsequent lactation)
[c]Treatment by body condition score (BSC) interaction reported, where thin: BCS ≤ 3.0, fair: BCS 3.25 to 3.75, fat: BCS ≥ 4.0 at 3 wk prior to calving
[d]2 trials reported

on milk yield or milk components were detected (34). Lasalocid in the ration at 360 mg/d decreased milk fat percent and increased protein percent, but had no significant effect on milk yield in a small study involving 12 rumen-cannulated cows (35). These 3 studies using lasalocid all suffered from a lack of power, due to the small sample size and large variance in milk yield among cows. Erasmus (19) evaluated the effects of 2 treatment groups of 10 ppm (concentration in mg/kg total food) and 20 ppm of monensin, fed 4 wk prepartum until 12 wk postpartum, in 60 multiparous cows in South Africa. Significant increases in milk production of over 3 kg/d were observed compared with untreated controls; however, no significant effects of monensin were noted for milk fat or milk protein percent. In a subsequent trial conducted in the United States that was designed to evaluate the impact of monensin fed prepartum and continuing through early lactation, no effect of treatment on milk yield or milk composition was observed (20). This project involved 47 Holstein cows and evaluated 3 levels of monensin (150, 300, or 450 mg/d), starting at 2 to 4 wk prepartum and fed until 84 d postpartum. The last 2 studies evaluated the impact of monensin on milk yield in early lactation. Few studies have assessed ionophore use throughout lactation. A study conducted in the United Kingdom evaluated the same levels of monensin (150, 300, or 450 mg/d) fed to 60 multiparous cows, commencing during the 6th wk of lactation (36). Monensin was associated with a non-significant decrease in feed intake and a significant increase in milk yield of 2.8 kg/d and 2.5 kg/d for the 150 mg and 300 mg monensin levels, respectively. Both milk fat and milk protein yields were reduced and the effect seemed to increase with the higher levels of monensin in the feed. This suggests a possible linear dose effect for monensin on milk fat percentage. A follow-up to this study showed that monensin fed through the dry period and into the subsequent lactation continued to exert similar milk production increases and milk fat percentage decreases (37). Erasmus et al (38) recently conducted a study in South Africa in which 60 dairy cows were randomly assigned to either 0, 10, or 20 ppm of lasalocid commencing 2 wk prior to calving through to week 17 of lactation. These authors concluded that

lasalocid had no impact on milk yield or milk components but decreased dry matter intake in a linear fashion. Therefore, they suggested that the use of lasalocid results in more efficient nutrient utlization. In the first of 2 trials, Van Der Werf et al (39) randomly assigned 64 Dutch Friesian cows to either no treatment or monensin at doses of 150, 300, and 450 mg/cow/d. They reported no significant effects of monensin on milk production but found a significant decrease in milk fat percentage associated with monensin treatment at 450 mg/cow/d. In the second trial trial, involving 58 Holstein and 22 Jersey cows, the authors observed increases in milk production but no effect on milk fat percentage for cows treated with monensin at 300 mg/cow/d. The authors reported better production responses to monensin in Holstein than in Jersey cows and in Holsteins of higher genetic merit. Interactions of ionophore treatment with breed or genetic merit have not been noted in previous studies; thus, these potential effects need further investigation.

A summary of the reported effects of ionophores on milk production and milk components in lactating dairy cattle is presented in Table 2.

Reproduction

There are now several reports of monensin's effect on reproductive performance in dairy cattle. An improvement in energy supply through increased propionate might be beneficial to improving reproductive performance. However, in 3 large studies, no significant effects of monensin were discovered for the interval from calving to first insemination, pregnancy rates, or days from calving to pregnancy (22,29,31). In a Canadian study, monensin CRC treatment had no significant impact on any measures of reproduction (24).

Conclusions

Lactating dairy cattle may benefit from ionophores in several ways. The literature strongly supports that monensin administered precalving has positive effects on energy metabolism in early lactation. These effects include a reduction in circulating ketone body concentrations and an increase in serum glucose. In addition, administration of the monensin CRC has been shown to reduce the incidence and duration of subclinical ketosis, and the risk of several periparturient diseases, including clinical ketosis and abomasal displacement. It appears that improved health would be the primary benefit of monensin used in early lactation. Although studies in Australia indicate that monensin reduces the risk of bloat, and studies in beef cattle suggest that monensin may be helpful in managing rumen acidosis, further work needs to done on the effects of monensin on rumen health in dairy cattle. In addition, the utility of lasalocid needs to be more completely assessed before its use in dairy rations can be recommended.

Studies included in this literature review indicate that monensin causes an increase in milk production of about 1 kg/d. Monensin tends to cause depression of milk fat, which may result in decreased milk fat yield. This effect appears to be dependent on dose and, possibly, other factors, such as stage of lactation and diet. Additional research is required to better ascertain the effect of monensin on milk production and components throughout lactation and in different dairy rations.

The use of monensin in dairy cattle appears to have many applications; thus, implementation strategies will vary with each farm and depend on the dairy producer's goals. Based on the current literature, monensin will be helpful in reducing the incidence of subclinical ketosis and other associated periparturient diseases, when treatment commences a few weeks precalving and extends toward peak lactation. Monensin will be of particular benefit to moderately and overly conditioned cows.

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BOOK REVIEW

COMPTE RENDU DE LIVRE

Boyer TH. ***Essentials of Reptiles: A Guide for Practitioners.*** Lakewood, Colorado, American Animal Hospital Association Press. 1998, 248 pp. ISBN 0-941451-65-8. US$ 46.95.

Essentials of Reptiles was written by a veterinary practitioner who is the most readable of authors on the husbandry and veterinary care of pet reptiles. This is a second and expanded wire-bound edition of his *Guide to Reptilian Husbandry and Care.*

The book is packed with valuable information for the veterinary practitioner on the medical and surgical management of some reptiles. It is comprised of chapters on General Information (biology and husbandry), Turtles and Tortoises, Green Iguanas, Leopard Geckos, Snakes, Caimans, and Anesthesia. Each chapter considers care and husbandry, diet, diagnostic and therapeutic procedures, and diseases. The text is indexed and well referenced for those seeking information both within the book and elsewhere. The list of citations is extensive (over 300). The book starts with an excellent review of other texts and information available on reptilian medicine, and ends with a formulary and a series of color plates featuring a selection of medical conditions.

This is a relatively inexpensive and very plain textbook, but it contains more valuable information than many glossier publications. The author's practical insights and extensive experience are presented simply and with lucidity. It is not a comprehensive text on reptile medicine and gives rather uneven treatment to the different reptile groups and topics. The chapter on leopard geckos should be taken as a model for other insectivorous species, as should the one on green iguanas for other herbivores. There seems to be little logic to the order of the sections: for instance, cystic calculi, neonatal iguanas, respiratory problems, and bloat are found consecutively. The color photographs are clumped at the end of the book and the captions for them are printed in the preceding pages, making them altogether less useful, although they are referenced in the text.

However, overall, this is a very readable and useful book, covering the majority of the medical conditions likely to be encountered in reptilian practice. With its strong slant on clinical practice, it presents the essentials of reptilian medicine, just as the title suggests.

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